makes modern bipolar devices more appealing and, to our advice, the real breakthrough in the arena of AF surgery. Such a characteristic is actually the greatest aspect of modern bipolar devices since, by clearing out the convective cooling of circulating blood on the target area, it allows an effective and reproducible ablation of a double layer of atrial wall epicardially, on the beating heart.

This notwithstanding, we respectfully acknowledge Patwardhan's contribution to innovation in this field. We express our empathy with his feelings at not being quoted in the context of bipolar RF. It is the same feeling we experience every time an author writes about epicardial ablation (be it bipolar or not) without mentioning our initiating clinical report[4] or when we realize that most authors reporting on surgical ablation—including Patwardhan in his later experience[2]—describe administering amiodarone prophylactically, but nobody seems to be aware that such pharmacologic strategy was first proposed for AF surgery by our group.[4,5]

We tend to blame the omission on a poor "conceptual copyright" protection policy on our side rather than suspecting the scientific accuracy of highly respected colleagues with whom we share the passion for finding new solutions to challenging clinical situations.

Stefano Benussi, MD, PhD
Simona Nascimbene, MD
Ottavio Alfieri, MD
Division of Cardiac Surgery
S Raffaele University Hospital
Milan, Italy

Stefano Benussi reports consulting fees from Atricure and Estech and lecture fees from St Jude Medical, Medtronic, and Cryocath.

References

1. Patwardhan AM, Dave HH, Tamhane AA, Pandit SP, Dalvi BV, Golam K, et al. Intraoperative radiofrequency microbipolar coagulation to replace incisions of maze III procedure for correcting atrial fibrillation in patients with rheumatic valvular disease. *Eur J Cardiothorac Surg.* 1997;12:627-33.
2. Lad VS, Patwardhan AM. Maze III replication using radiofrequency microbipolar coagulation. *Heart Lung Circ.* 2004;13:139-44.
3. Benussi S, Nascimbene S, Calori G, Denti P, Ziskind Z, Kassem S, et al. Surgical ablation of atrial fibrillation with a novel bipolar radiofrequency device. *J Thorac Cardiovasc Surg.* 2005;130:491-7.
4. Benussi S, Pappone C, Nascimbene S, Oreto, Caldarola A, Stefano PL, et al. A simple way to treat atrial fibrillation during mitral valve surgery: the epicardial radiofrequency approach. *Eur J Cardiothorac Surg.* 2000;17:524-9.
5. Benussi S, Nascimbene S, Alfieri O. Reply to Veloso. *Eur J Cardiothorac Surg.* 2001;19:233-4.

doi:10.1016/j.jtcvs.2007.01.057

Immediate surgery in aortic dissection with cerebral malperfusion

To the Editor:

We congratulate Estrera and colleagues[1] and Urbanski and coworkers[2] on their impressive results with immediate aortic surgery for acute type A dissection with cerebral malperfusion. However, we would like to add specific observations, according to our experience with this complex issue.[3]

Estrera and associates[1] report a case series of 16 patients with acute aortic dissection and stroke, outlining a correlation between the time interval before aortic repair and outcome. Specifically, neurologic status never worsened, whereas recovery or improvement was observed in 80% of patients operated on within 10 hours. Surgery was denied in 1 patient with "neurologic devastation," defined as coma with a Glasgow score of 5 or less. This definition appears somewhat arbitrary and refers to a single patient in this series, which is insufficient to draw specific recommendations. We previously reported on 5 comatose patients selected for immediate repair on the basis of hemodynamic stability, preserved pupillary reactivity, and coma duration of 12 hours or less, with encouraging results.[3] Median Glasgow coma score was 5.5, whereas all patients showed an eye response score of 1. Similarly, all documented strokes in our series were right-sided, which likely indicates the same underlying pathophysiology, namely, malperfusion with dynamic flow patterns and prevalent compression of the innominate artery true lumen. In this respect, we pointed out the unsuitability of the Glasgow coma score, originally developed for the evaluation of neurologic trauma, to stratify these patients. Importantly, prompt aortic repair has been successfully reported with a score of 3,[4] whereas neurologic recovery has been reported with extra-anatomic revascularization and delayed aortic repair more than 2 decades ago.[5] Furthermore, the Rankin score also failed to correlate with outcome in this series, suggesting that pathophysiologic mechanisms are different than those involved in acute occlusion causing traditional stroke.

Urbanski and colleagues[2] introduced the left common carotid as a systemic perfusion route for arterial return, with excellent results in 100 patients, including 27 with acute dissection, 4 of whom had cerebral malperfusion. In view of the prevalence of right-sided arch vessel malperfusion in the case of an acutely dissected aorta and cerebral injury, this approach seems particularly appealing in this context, especially if cannulation via a prosthetic vascular graft is established distant from the arch, thus avoiding dissected segments. Furthermore, this strategy reduces embolic hazards and greatly simplifies the implementation of selective antegrade brain perfusion. However, there is also a rationale for additional innominate artery perfusion after intraluminal arch inspection, which requires only an insignificant period of total circulatory arrest. In our opinion, we would hesitate to not provide selective antegrade perfusion to the most severely ischemic territory.

In summary, available reports indicate the importance of a nondelayed attitude for a satisfactory outcome in selected patients with acute type A aortic dissection and cerebral malperfusion. Current scores appear unsuitable to stratify the severity of neurologic injury in this specific context and should be viewed cautiously when the intent is to define irreversible "neurologic devastation," particularly in stable patients with preserved brain stem reflexes.

Marco Pocar, MD, PhD
Davide Passolunghi, MD
Andrea Moneta, MD
Francesco Donatelli, MD
Cattedra di Cardiochirurgia
Università degli Studi di Milano
IRCCS MultiMedica
Milano, Italy

References

1. Estrera AL, Garami Z, Miller CC, Porat EE, Achouh PE, Dhareshwar J, et al. Acute type A aortic dissection complicated by stroke: can immediate repair be performed safely? *J Thorac Cardiovasc Surg.* 2006;132:1404-8.
2. Urbanski PP, Lenos A, Lindemann Y, Weignag E, Zacher M, Diegeler A. Carotid artery cannulation in aortic surgery. *J Thorac Cardiovasc Surg.* 2006;132:1398-403.

3. Pocar M, Passolunghi D, Moneta A, Mattioli R, Donatelli F. Coma might not preclude emergency operation in acute aortic dissection. *Ann Thorac Surg.* 2006;81:1348-51.
4. Fujii H. Is coma an absolute contraindication for emergency central aortic operation? *J Thorac Cardiovasc Surg.* 2004;128:749-50.
5. Walterbusch G, Oelert H, Borst HG. Restoration of cerebral blood flow by extraanatomic bypass in acute aortic dissection. *Thorac Cardiovasc Surg.* 1984;32:381-2.

doi:10.1016/j.jtcvs.2007.01.075

Reply to the Editor:

We thank Pocar and colleagues for their interest in our article regarding surgery for acute type A aortic dissection in the setting of stroke.[1] We also acknowledge their recent report about repairing acute type A aortic dissection in patients with coma.[2] As was pointed out in their report, the use of the Glasgow Coma Scale (GCS) to exclude patients for type A aortic repair was not relevant. Specifically, they noted that all 5 of their patients would have been considered severely brain injured (GCS $<$ 8) based on the GCS alone. We do appreciate and concur with this point and also congratulate them for them for their results in this very difficult subgroup of patients.

Although the GCS, the National Institutes of Health Stroke Scale (NIHSS), and Rankin score for that matter may not be completely applicable in the setting of acute type A aortic dissection, we wanted to analyze these patients with objective criteria. We do believe that the NIHSS and the Rankin score may be helpful, although this study was not powered to demonstrate this. Because we recommended that operative repair was indicated in patients *without* "neurologic devastation" or coma, we attempted to provide some objective criteria for this condition, hence the use of these scales and scores. Regarding the 1 nonoperated patient who was considered neurologically devastated, we acknowledge that one cannot derive any conclusions in relation to defining neurologic devastation. In fact, it was the appearance of the patient's computed tomography scan of the head, which showed bilateral massive infarction, that ultimately led to his nonoperative course.

Prior to the results of this study, we maintained that stroke was a relative contraindication to immediate repair for acute type A aortic dissection. We have since modified our approach and have become more aggressive in repairing acute type A aortic dissection in patients with stroke. Our experience in patients with coma, however, has been limited and thus we continue to maintain a selective approach in patients with coma and neurologic devastation. We admit that GCS is not a good measure of coma or neurologic devastation, and radiographic evaluation, in the hemodynamically stable patient, may be more helpful. How coma and neurologic devastation are determined and whether or not surgery is performed, however, should be left ultimately to the neurologist and operating surgeon, respectively.

Anthony L. Estrera, MD
Hazim J. Safi, MD
University of Texas Medical School
Houston, TX

References

1. Estrera AL, Garami Z, Miller CC, et al. Acute type A aortic dissection complicated by stroke: can immediate repair be performed safely? *J Thorac Cardiovasc Surg.* 2006;132:1404-8.
2. Pocar M, Passolunghi D, Moneta A, et al. Coma might not preclude emergency operation in acute aortic dissection. *Ann Thorac Surg.* 2006;81:1348-51.

doi:10.1016/j.jtcvs.2007.02.024

High-dose atorvastatin is associated with impaired myocardial angiogenesis in response to vascular endothelial growth factor in hypercholesterolemic swine: Relevance to the human situation?

To the Editor:

We read with great interest the article by Boodhwani and colleagues[1] regarding the impact of high-dose statin therapy on vascular endothelial growth factor-A (VEGF-A)–induced myocardial angiogenesis in a hypercholesterolemic pig model of chronic ischemia. This article demonstrated that collateral-dependent myocardial perfusion remained impaired in hypercholesterolemic and atorvastatin-treated pigs in response to additional treatment with VEGF-A relative to a normocholesterolemic control group. It concluded that a high-dose statin therapy was not associated with improved myocardial neovascularization. In that study, however, the hypercholesterolemic pigs were treated with an atorvastatin dosage of 3 mg/(kg $\cdot$ d), which exceeds the maximal possible dosage in patients (80 mg/d) by a factor of about 3.

To achieve at least moderate cholesterol lowering, pigs need to be treated with high-dose statins because of lesser lipid-lowering potency and efficacy in pigs relative to human beings.[2] One must remain aware, however, of the increased risk of adverse events with extremely high statin dosages. In their article, Boodhwani and colleagues[1] described a clearly decreased capillary endothelial cell density in the ischemic territory in the hypercholesterolemic and atorvastatin-treated pigs relative to the normocholesterolemic pigs and even the untreated hypercholesterolemic animals. To exclude the possibility that the hypercholesterolemic and atorvastatin-treated pigs suffered potential toxic (cellular) side effects of atorvastatin that might explain the neovascularization impairment, the investigators should have incorporated a control group of normocholesterolemic pigs treated with the same atorvastatin dosage. Moreover, treatment of the hypercholesterolemic pigs with lower atorvastatin dosages, more relevant to the human situation, might demonstrate whether there are potential dose-dependent toxic cellular side effects. Indeed, a recently published article by Chade and associates[3] indicates that statins used in an intermediate (human) dosage in pigs do lead to a stimulation of arteriogenesis. Furthermore, a retrospective clinical study supports the view that intensified statin treatment is associated with an improved arteriogenic response in human beings.[4]

Another piece of evidence for statin overdose in the hypercholesterolemic pigs of Boodhwani and colleagues[1] might be the observation of prolonged *Akt* activation in the atorvastatin-treated pigs. Therefore, the *Akt* downstream signal transduction pathways in endothelial cells might not any longer be able to respond adequately to other pathophysiologic stimuli. Indeed, it has been demonstrated that a prolonged *Akt* activation is associated with detrimental cardiac effects in an ischemic mouse model,[5] as was even cited by Boodhwani and colleagues[1] in their article.

Taken together, we believe that it is not appropriate to compare directly the arteriogenic effects of high-dose statin treatment in pigs and human beings in light of the approximately 3-fold higher statin dosage used in pigs.